data when we derive diagnostic conclusions and determine subsequent therapy.

People come to doctors when they feel something is wrong. We try to explain their complaints in terms of "disease"—that is, as an abnormality in a physiologic system—but we tend to ignore the "illness"—that is, the general discomfort of the patient. It was Eric Cassell who most recently drew our attention to this point, which was nicely highlighted by the two cases cited by Haas et al in their article. Each of the patients had proven EIB after careful, detailed study, but both of them were such remarkable performers physically and their preliminary pulmonary function tests were so near normal that their basic disease was overlooked because their "illness" was assigned too little importance. To maintain our credibility not only with our patients but with ourselves and our self-image as healers, we must be very careful about undervaluing the complaints made by our patients.

In summary, I wish to be clear: In general, patients' statements concerning their state of health are more accurate than the machines we use to measure the patients' physiologic and biochemical functions, and the data generated by a good history must be assigned a correspondingly high value. We must intensify our research into the physiologic and biochemical bases for patients' complaints so that we have more reliable data to explain, but not necessarily to contradict, what our patients tell us. Finally, we must develop scientific methods for better quantifying and discriminating the importance of what our patients have to say. I believe that this effort takes on added importance as our scientific capability becomes more and more sophisticated.

Drs Haas, Axen, Salazar-Schicchi, the challenges implicit and explicit in your paper leave me breathless!

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The Effect of Theophylline on Sleep in Normal Subjects

Until recently, the effect of bronchodilators on sleep architecture has been relatively unstudied. The assumption has been that medications that relieve airway obstruction during the daytime should do so during the night as long as plasma levels are maintained. However, reestablishing airflow at night while remaining asleep is quite distinct from restoring normal airway function during wakefulness. It is well known that minor changes in various respiratory stimuli, such as hypercapnia, hypoxia, airway occlusion, and pulmonary irritation, produce arousals and disruption of the sleep pattern. Establishing, therefore, that a patient sleeps normally provides good indirect evidence that overall airway function is being adequately maintained.

In general, sleep architecture can be viewed in terms of "quantity," as determined by the total sleep time and efficiency (actual recorded total sleep time divided by total time in bed), and "quality," as assessed by fragmentation due to small arousals lasting 10 to 20 s or shifts to lighter stages. Clinically significant disruption in either the quantity or quality of sleep results in the sensation of daytime fatigue and sleepiness. Since patients with underlying lung disease are known to already suffer from disrupted sleep, it is useful to distinguish any potential deleterious effect that a particular medication might have on overall sleep architecture apart from its beneficial effect on airway function.

In this issue of Chest (see page 193), Kaplan and coworkers report the effects of theophylline on sleep architecture in ten healthy young men in a double-blind cross-over design. They found that theophylline decreased total sleep time by approximately a half hour (decreased quantity) as well as increased the number of arousals (decreased quality) by four episodes per hour. Otherwise, sleep efficiency, the distribution of stages of sleep, and sleep onset times were unaltered. These findings are in slight contradistinction to those of another recently published study of similar design, which showed no effect of theophylline on either sleep architecture or daytime cognitive performance. Since both studies were almost identical in design, it is hard to explain the differences in total sleep time other than to note that the study by Fitzpatrick et al contained a selection bias due to symptomatic dropout and a longer period of adaptation to theophylline.

In fact, both studies are giving us similar clinical information. That is, theophylline probably does not clinically alter sleep architecture enough to cause clinical daytime dysfunction. This conclusion is based on recent sleep deprivation work demonstrating that less than 1 to 2 h of chronic reduction in total sleep time will not likely affect either symptomatic or objective measurements of daytime sleepiness. Likewise, the minimal increase in hourly arousals in the study by Kaplan et al will probably not significantly alter daytime function since we know that older normal individuals, who have demonstrated much higher arousal rates, did not show clinically significant alter-
ations in daytime sleepiness.11,12 Finally, after adjusting for the age differences between the subjects in the studies by Kaplan et al and Fitzpatrick et al, it can be seen that the distribution of sleep architecture, the sleep onset latency, the sleep efficiency, and wake after sleep onset were almost identical, suggesting that theophylline did not directly alter the fundamental mechanisms that determine the specific stages of sleep.

These two studies provide useful new information in evaluating the effectiveness of treatment of obstructive airway disease during sleep. For example, 10 years ago Montplaisir et al13 noted a marked increase in total sleep time of almost 2 h when a group of asthmatic patients were treated. Although the precise regimen was not specified, the improvement most likely was due to better management of nocturnal airway function. Moreover, from the few studies that have examined sleep in patients with chronic obstructive and restrictive disease, it is clear that there are typically striking reductions in the quantity of sleep, with total sleep times often averaging 200 to 250 min. Thus, rather than worrying too much about the minimal effects of theophylline on sleep architecture in normal individuals, it would now seem more useful to know whether patients whose sleep is so severely disrupted can be helped by pharmacologic or other means.

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Abstracts from European Respiratory Journal

A New Service for our Readership

The European Respiratory Journal is the official journal of the European Respiratory Society. It was formed from a merger of the Bulletin European de Physiopathologie Respiratoire and the European Journal of Respiratory Diseases. This periodical is a peer-reviewed scholarly journal which publishes original investigations from researchers throughout the world. The primary emphasis, however, is on clinical research in European countries.

We believe that reports of these investigations, both basic and clinical, may be of considerable interest to physicians in North America, South America, and the Orient as well. Therefore, in cooperation with Professor Paul Vermeire, Editor-in-Chief of the European Respiratory Journal, we would like to offer the readers of Chest selected abstracts from current issues of Professor Vermeire’s periodical. In this issue of Chest, we inaugurate this new section. This month, and in subsequent issues, the exact location of the abstracts will be shown on the contents page.

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